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Cell Injury and Interstitial Inflammation in Rat Lung after Inhalation of Ozone and Urban Particulates

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Coexposure of the lung to urban dust along with ozone appears to potentiate ozone-induced injury. This conclusion was derived from whole-lung studies involving tissue and lavaged cells, but we now speculate that the injury and inflammatory response at the main site of reactivity, the bronchoalveolar duct region, is underestimated by such whole-lung studies. We exposed rats to ozone at 0.8 ppm and urban particulates (EHC93) at 50 mg/m³ for 4 h. Animals were killed 33 h later with tritiated thymidine (³HT) injected 1.5 h before death. Lungs were fixed by vascular perfusion to avoid disturbing any epithelial cell changes or local inflammation and edema in the air spaces. Tissue was embedded from central and peripheral areas of the lung, then counts of labeled cells, polymorphonuclear leukocytes (PMN), and macrophages (MAC) were made separately on methacrylate sections. The results showed that epithelial cell injury and regeneration (% of ³HT-labeled cells) was greatest in the ozone plus dust group, and was three times higher in periductal areas than in whole-lung counts. Although some increase in inflammatory cells in the air spaces was found in the coexposure group, much higher numbers of PMN and MAC were counted in the lung tissue compartment, and counts were significantly higher than those found after ozone or dust alone. Values from the latter groups were also higher than those from air controls or samples of distal lung taken from any inhalation group. The results demonstrate that inhalation of an urban dust at a level that causes few lung effects when inhaled alone can potentiate ozone toxicity, particularly in the vicinity of the alveolar duct, where the accumulation of interstitial inflammatory cells may be an important factor in the development of any subsequent pathologic changes.

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